

Cardiovascular Toxicity of Primary Combustion Particles: Linking Adverse Health Effects to Sources

Experimental Toxicology Division
NHEERL

Travis Knuckles¹, Richard Jaskot², Judy Richards², Elizabeth Murphy³, James Russell⁴, and Kevin Dreher²
¹North Carolina State University, ²US EPA, ³NIH, NIEHS, and ⁴University of Alberta, Canada

Environmental Issue: The overall weight of epidemiological evidence has indicated a significant association between ambient air particulate matter (PM) exposure and adverse health effects with susceptible sub-populations. Insight into the biological plausibility (PM hazard identification, mechanisms of injury, and susceptibility) for the observed epidemiological associations with PM is still lacking and remains a critical area of research for PM risk assessment and management. Epidemiological studies have tried to determine the contribution of fine PM (PM_{2.5}), derived from anthropogenic processes such as combustion of fossil fuels and atmospheric transformations, and coarse (PM_{10-2.5}), derived from natural emission sources, have on PM-associated acute mortality and morbidity. Schwettje et al. reported that PM_{2.5} particles not coarse particles were associated with PM acute mortality and their finding has been confirmed in a re-analysis of this data by Klemm and Masson (1, 2). Schwartz et al. and Pope et al. reported that PM_{10-2.5} was associated with PM mortality (3, 4). Of the various pollutants examined by Fairley, the strongest association with mortality occurred with PM_{2.5} (5). More recent epidemiological studies have also investigated the relative importance of fine versus coarse air particle pollution in PM-associated acute mortality. Seven studies (2, 6 - 11) have reported greater correlations between PM_{2.5} and acute PM-associated mortality, whereas 4 studies found greater correlations between PM_{10-2.5} and acute PM-associated mortality (12 - 15). Finally, two studies have shown PM-associated morbidity correlated better with PM_{2.5} than for levels of PM_{10-2.5} (6, 16). These conflicting results suggest that geographical location, population demographics, and co-pollutant mixture effects may affect the ability of epidemiological approaches to unequivocally identify cause PM particles and their sources. It is clear that controlled toxicology studies are critically needed in order to validate epidemiological based studies that have tried to link PM health effects with specific PM emission sources and atmospheric processes.

References:
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IN VIVO COMBUSTION PM CARDIOTOXICOLOGY

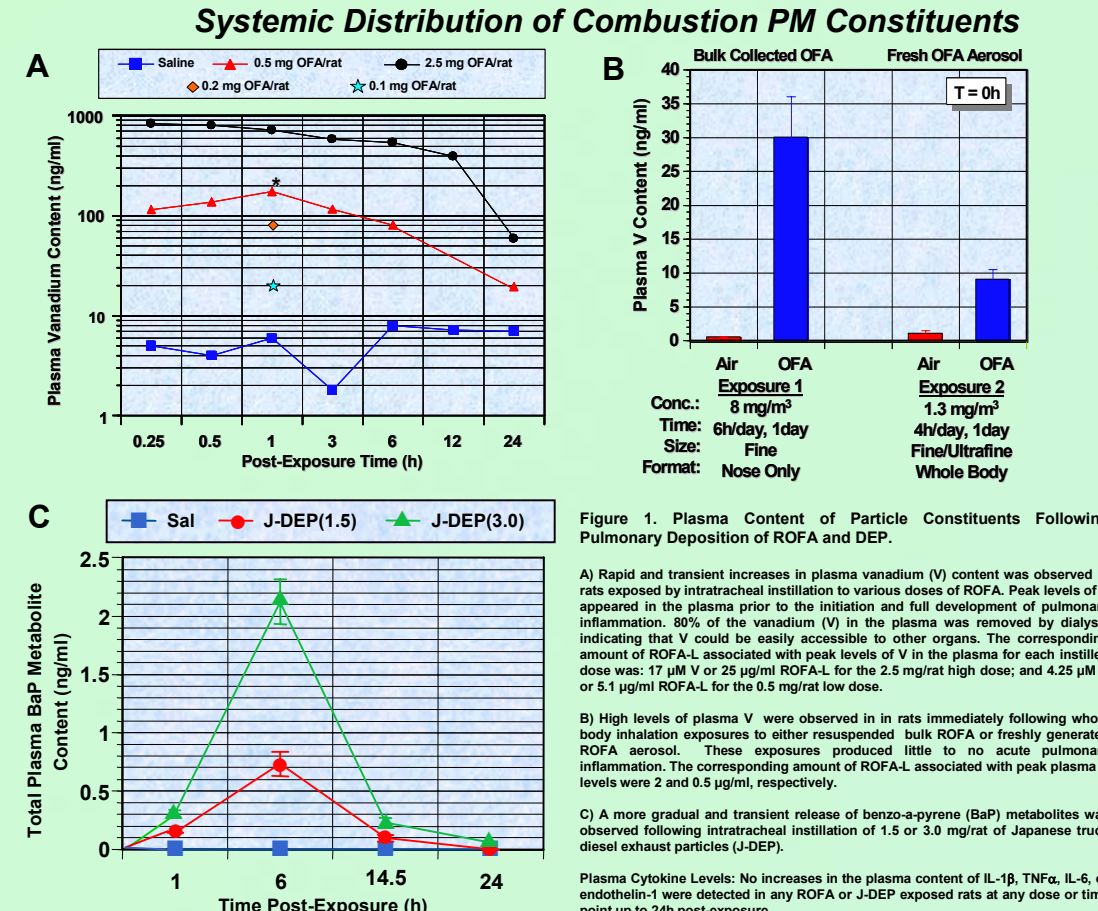


Figure 2. Oil Combustion PM Activation of Cardiac MAP Kinase Cell Signaling. Protein extracts obtained from hearts recovered from rats following intratracheal instillation of either saline or ROFA (2.5 mg/ml) were examined for alterations in cardiac intracellular signaling. Pulmonary exposure to ROFA led to a rapid and transient activation of ERK1/2 MAP kinase. Similar results were obtained for P38 (data not shown). No effect on JNK/MAPK was observed (data not shown). Activation of these two MAP kinases occurred prior to full development of ROFA-induced lung injury.

Cardiac Intracellular Signaling Pathway Activation
ERK1/2 Extract Activity

